BLOOD GLUCOSE, CARDIAC AND SKELETAL MUSCLE GLYCOGEN, ELECTRO-CARDIOGRAM AND HAEMATOLOGICAL CHANGES IN ACUTE HEAT STRESS— MODIFICATION BY PHYSOSTIGMINE AND ATROPINE

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Summary: Heat stress produced hyperglycaemia, a decrease in myocardial and skeletal muscle glycogen levels, eosinopenia and an increase in heart rate. Physostigmine alone produced a fall in the blood glucose and myocardial and skeletal muscle glycogen contents, eosinopenia and a decrease in heart rate. Heat stress and physostigmine produced hypoglycaemia, a decrease in myocardial glycogen content, an increase in skeletal muscle glycogen content and eosinopenia. Atropine alone produced hypoglycaemia, a decrease in myocardial glycogen content, increase in skeletal muscle glycogen content, eosinopenia and an increase in heart rate. Heat stress and atropine produced hypoglycaemia decrease in myocardial and skeletal muscle glycogen contents and eosinopenia.

Key words: heat stress, glucose, glycogen, ECG, haematology, physostigmine, atropine

INTRODUCTION

Stress produces biochemical, histological, haematological, and functional changes in the body (17). The effects of acute heat stress on blood glucose, cardiac and skeletal muscle glycogen, ECG and haematology are presented in this communication. Such a study seems warranted because most of the Indians live in tropical climate which varies in humidity, Further, hot environment is a problem in many industries and in deep mining. The literature on the role of parasympathetic system in heat stress is scant. Our previous work on antiadrenergic agents (5, 18, 19) prompted us to assess the role in heat stress of parasympathetic system.

MATERIALS AND METHODS

Albino rats of either sex (80-120 *g*) were used. They were maintained at a temperature range of 30°C—32°C, and had ad libitum access to food and water.

The animals were divided into 6 groups employing eight to ten animals in each group. Group I animals served as negative control, and were not subjected to any stress. Group II served as positive control; this group of animals were subjected to heat stress by the modified method of Mahfouz and Ezz (10). The animals were exposed to 40°C±0.5°C for one hr, by keeping them in a cylindrical glass jar 1 foot high and 9 inches in diameter, surrounded by water, the temperature of which was maintained at 41°C±0.5°C. The jar was kept open to prevent

anoxia and upto 4 rats were kept at a time in the jar. The temperature of the air in the glass jar 1 inch above the bottom was 40°C. Group III animals were given 0.1 *mg/kg* of physostigmine sulphate (im) thrice during 24 hr at equally spaced intervals and were not subjected to any stress. Animals of group IV received atropine sulphate in a dose of 1.0 *mg/kg* (im) thrice during 24 hr at equally spaced intervals and were not subjected to any stress. Rats of group V were given 0.1 *mg/kg* of physostigmine (im) in a single dose 30 min. before subjecting them to heat stress. Animals of group VI were given atropine 1.0 *mg/kg* (im) in a single dose 30 min. before subjecting them to heat stress.

Blood was collected directly from the heart of the rat, killed by ether anaesthesia, 30 min after the individual procedure. Heart tissue (tip of the ventricles) and gastrocnemius muscle were collected for glycogen estimation. Blood glucose was estimated by the Somogyi-Nelson method (11, 21) myocardial and skeletal muscle glycogen contents by the Kemp-Kits Van Heijningen method (8), clotting time by the Lee-White method (9) and eosinophil count by Pilot method (12). Lead II of E.C.G. (by fixing the leads with needles), was recorded in unanaesthetized rats with Gras inkwriting oscillograph just before and after heat stress.

RESULTS

The data are summarised in Table I.

TABLE I: Heat stress and some body functions.

Group	*Blood glucose mg/100 ml* *mean ± S.E.*	*Heart glycogen mg/100 g of tissue* *mean ± S.E.*	*Skeletal muscle glycogen mg/100g tissue* *mean ± S.E.*	*Blood clotting time in min.* *tissue* *mean ± S.E.*	*Eosinophils/cmm of blood* *mean ± S.E.*
Negative control	98 ± 0.63 (9)	161 ± 8.7 (9)	158 ± 8.7 (8)	1 ± 0.33 (8)	432 ± 2.8 (8)
Heat stress	109 ± 3.7** (8)	99 ± 4.1* (8)	139 ± 4.5** (8)	2 ± 0.28 (8)	249 ± 5.6* (8)
Physostigmine alone	71 ± 1.3* (10)	57 ± 4.7* (8)	98 ± 5.5* (8)	2 ± 0.25 (8)	285 ± 4.7* (8)
Atropine alone	54 ± 2.12* (10)	84 ± 4.8* (8)	249 ± 9.5* (8)	1 ± 0.35 (8)	298 ± 3.3* (10)
Physostigmine followed by heat stress	68 ± 5.2* (8)	58 ± 2.4* (8)	254 ± 13.4* (8)	1 ± 0.28 (8)	324 ± 4.6* (8)
Atropine followed by heat stress	65 ± 4.8* (8)	22 ± 2.1* (8)	120 ± 3.1 (8)	1 ± 0.117 (9)	299 ± 3.4* (9)

*P<0.001, **P<0.05, ***P<0.01 Compared to control values (t test).
Figures in parentheses indicate the number of animals.

Blood glucose: There was a significant rise in blood glucose level in animals subjected to heat stress. Both physostigmine and atropine lowered the level, atropine being more effective. In animals treated with physostigmine or atropine followed by heat stress the level was lower than in negative controls.

Myocardial glycogen content: Heat stress or physostigmine alone or atropine alone reduced myocardial glycogen level. Exposure to heat stress following physostigmine or atropine treatment also produced decrease in the level, the effect being more pronounced in atropine pretreated rats.

Skeletal muscle glycogen content: Heat stress lowered the skeletal muscle glycogen content. Physostigmine alone lowered it but increased it when combined with heat stress. On the other hand, atropoine alone raised it but reduced it when combined with heat stress.

Clotting time: Insignificant increase in blood clotting time was observed in animals subjected to heat stress. No significant change was seen in animals treated with physostigmine or atropine alone or in combination with heat stress.

Eosinophil count: A marked eosinopenia was observed in animals subjected to heat stress; similarly physostigmine or atropine treatment alone or in combinations with heat stress produced marked eosinopenia.

Electrocardiogram (ECG): Group II animals exposed to heat stress showed a mean increase in heart rate of 27 beats/min. Pretreatment with physostigmine caused a mean fall of 23 bets/min, while atropine pretreatment caused a mean rise of 20 beats/min. Exposure of animals pretreated with physostigmine or atropine to heat stress produced mean increases in heart rate of 28 beats/min and 57 beats/min respectively.

In heat stress, no other abnormality was observed except an increased QT interval and bifid T wave in 1 of the 6 animals. In physostigmine-treated animals flattened P wave in 1, increased QT interval in 3 and bifid T wave in 2 of the 7 animals was observed. No change except increased QT interval in 4 of the 8 animals was observed in atropine-treated rats. Exposure to heat stress following treatment with physostigmine or atropine produced an increased QT interval in 4 of the 8 and 2 of the 8 animals respectively.

DISCUSSION

Hyperglycaemia that occurs during hyperthermia (4,17) may be due to increased activity of adrenergic system, which leads to increased mobilization of glucose from the liver, since there is no hyperglycaemia during hyperthermia induced after adrenergic blockade (4). Further, hyperglycaemia is not due to haemoconcentration since the haematocrit and specific gravity of blood are relatively constant (4).

The present study also demonstrates the hyperglycaemic effect after heat stress. The hyperglycaemia could be due to release of adrenaline during stress. Physostigmine given alone

or given to stressed animals produced hypoglycaemia. An explanation for this could be th physostigmine causes increased adrenal medullary discharge of adrenaline (7) which inhibi cholinesterase activity (1, 2) resulting in an increased level of acetylcholine, the latter antagonizin the sympathetic activity in stressful situation to maintain the homeostatsis. However, an intriguin finding was that atropine alone or atropine following exposure to heat stress also lower blood sugar level.

A fall in cardiac glycogen occurs during alarm reaction of general adaptation syndrome (15 and during heat stress (17). Thd fall during heat stress has been explained as due to the endogenous release of catecholamines, since this effect is blocked by chlorpromazine. Sympathetic overactivity during stress increases the oxygen need and causes hypoxia which results in increased utilization of glycogen leading to a fall in its level (14). In the present study a fall in myocardial and skeletal muscle glycogen was noted during heat stress. In agreement with our earlier observations (18) physostigmine treatment also reduced the level. Physostigmine causes increased discharge of adrenaline from the adrenal medulla (7) and releases locally acting catecholamines from the heart (6). This could account for the depletion of glycogen. Atropine administration resulted in a reduced myocardial but raised skeletal muscle glycogen content. Physostigmine offered a complete protection against the skeletal muscle glycogen depleting effect of heat stress but not against the myocardial glycogen depleting effect.

In animals subjected to heat stress an insignificant increase in clotting time was observed with Lee and White method. A significant reduction in clotting time has been reported with the capillary tube method (13). The difference appears to be due to the different methods employed as it is known that temperature and size of the tube are important factors which affect clotting time.

Significant eosinopenia observed in the animals subjected to heat stress confirms the literature reports (13,22).

Stress produces variable changes in the electrocardiogram. An increased heart rate after heat stress observed in the present study is in accord with literature reports (5,3,20).

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